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Volume 234 APRIL 18, 1946 Number 16

CLINICAL AND LABORATORY STUDIES OF RELAPSING VIVAX MALARIA OF PACIFIC ORIGIN*

LIEUTENANT COLONEL HARRY H. GORDON, M.C., A.U.S., COLONEL ALEXANDER MARBLE, M.C., A.U.S.,
LIEUTENANT COLONEL STUART W. LIPPINCOTT, M.C., A.U.S.,
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CAPTAIN LESTER D. ELLERBROOK, SN.C., A.U.S.

SINCE members of the armed forces have been stationed in areas in which malaria is prevalent, many of them will experience symptoms of the disease, either as delayed primary attacks or as relapses, when suppressive treatment is discontinued on their return to nonendemic areas. It seems worth while, therefore, to present a brief summary of the clinical features and laboratory findings in 608 soldiers admitted from Pacific areas to the Malaria Section of the Harmon General Hospital from August, 1943, to March, 1945. Certain of the data have been presented in greater detail in recent articles.[1-4]

TYPE OF MALARIA

Although the patients came from areas in which falciparum malaria was endemic, and although many of them stated that some of their previous malarial attacks had been caused by *Plasmodium falciparum*, this organism was demonstrated in only 1 of 864 clinical attacks, and in this case it contributed to a mixed infection with *P. vivax*. This evidence strongly supports the statement that the use of quinacrine (Atabrine) in adequate doses completely cures falciparum malaria.[5] From the discussion that follows, it will be noted that the clinical features of vivax malaria observed in these patients differed considerably from those described in falciparum infections by Most and Meleney,[6] but it should be pointed out that extremely few of the patients had arrived in the United States directly from areas in which there was inadequate control of mosquitoes.

INFECTIOUSNESS

The infectiousness of patients with clinical attacks of malaria depends on the presence of gametocytes in the peripheral blood. Thin smears were examined for gametocytes in a total of 401 attacks. In 196 (49 per cent), they showed gametocytes. In 255 attacks, treatment was not begun for approximately forty-eight hours after the onset of the relapse. In only 41 attacks (16 per cent) did blood smears taken daily or twice daily show the development of two or more gametocytes per 100 white cells, a level at which it is considered worth while to attempt to infect mosquitoes for transmission tests.† Since prompt initiation of treatment quickly eradicated parasites (Table 1), it is obvious that there is extremely little danger of transmission of the disease if it is diagnosed promptly and if treatment is begun immediately after a positive smear is obtained. This does not in any way mitigate the need for adequate mosquito control. During a period of over one and a half years during which patients with malaria have been treated at this hospital, not a single patient has been admitted to the wards whose disease was considered to have been contracted here.

TABLE 1. *Effect of Quinacrine on Blood Smears and Fever.*

INTERVAL AFTER BEGINNING OF TREATMENT*	PERCENTAGE OF 321 PATIENTS WITH NEGATIVE SMEARS†	PERCENTAGE OF 434 PATIENTS WITH ORAL TEMPERATURES OF 99.2°F. OR LESS
days		
1	33	26
2	94	71
3	99	83
4	99	91

*Treatment of acutely ill patients was begun soon after admission to the ward, but only if both a positive smear and an oral temperature of 100°F. or higher were present.

†Only those patients are included who had a smear taken on each day.

CLINICAL FEATURES

The two essential clinical features of the disease as observed in this study were the relative mildness of the acute attack and its stubborn tendency to relapse.

*From the Army Service Forces, Eighth Service Command, Harmon General Hospital, Longview, Texas.

†The parasite counts in these patients were made by the staff of the Laboratory of the United States Public Health Service Imported Malaria Studies. The transmission tests referred to were carried out by this group to determine whether oversea strains of malaria were transmissible by means of infected, laboratory-bred American mosquitoes. The results of this study will be reported elsewhere.[7]

Characteristics of the Acute Attack

Less than 10 per cent of the patients showed any cerebral symptoms. The prompt subsidence of such symptoms with routine therapy led to the impression that these manifestations were such as would be found in any group of patients with high fever.

The infrequency of troublesome vomiting is evidenced by the fact that parenteral fluids had to be administered in less than 10 of the 864 attacks. Nausea and vomiting were present in about one fourth of the attacks, but with subsidence of the acute febrile phase under quinacrine therapy these symptoms gradually cleared.

There were prompt subsidence of fever and other symptoms and rapid disappearance of parasites from blood smears with the administration of quinacrine in customary doses of 0.2 gm. every six hours for five doses, followed by 0.1 gm. three times a day for six days until a total of 2.8 gm. had been given. Table 1 shows that on the second day after the beginning of quinacrine therapy, 94 per cent of the patients had negative smears and 71 per cent had a maximum oral temperature of 99.2°F. or less. Since a large majority of these patients had previously had several attacks of malaria, most of which had been treated with quinacrine, the prompt eradication of malarial parasites in these attacks indicated the continued effectiveness of the drug in the acute attack.

Interval Symptoms

Although the acute attacks of malaria were relatively mild and the symptoms promptly subsided with quinacrine therapy, many of the patients complained of easy fatigability, nervousness and dyspepsia between relapses. In the majority of cases these symptoms seemed to be strongly conditioned by factors such as exposure to combat and adverse living conditions in the Pacific areas or previous personality problems. Recurrent malaria added to the patients' difficulties but was not primarily responsible for them. The patients needed much reassurance that the ultimate prognosis for the complete cure of their malaria was good, since they had returned to areas in which the chance of reinfection was slight.

Data on Relapses

The marked tendency to relapse is indicated by the fact that of 287 patients with attacks treated with quinacrine at this hospital and followed either until a proved relapse occurred or at least sixty days had elapsed after treatment was completed, 193 (67 per cent) had relapses. As is evident in Table 2, neither the continued use of small doses of quinacrine for sixty days (compare Groups II and III with Group I) nor the use of pamaquin (Plasmochin) in the indicated dosage (compare Group II with Group III) had any significant effect on the ultimate rate of relapse.

In Table 3 it is seen that of all the relapses that occurred up to six or seven months after completion of treatment with quinacrine, 60 per cent took place within sixty days and 90 per cent within four months. The higher percentage of relapses in

TABLE 2. *Number of Relapses following Treatment with Quinacrine.**

	GROUP I	GROUP II	GROUP III	TOTALS
Number of patients....	124	86	77	287
Number of relapses†...	92	52	49‡	193‡
Percentage of relapses .	74	60	64	67

*All patients received 0.2 gm. of quinacrine every 6 hours for five doses, followed by 0.1 gm. three times a day for 6 days, until a total of 2.8 gm. had been given. At the completion of this therapy, the patients in Group I received no further treatment; patients in Group II received no treatment for 1 day, followed by 0.01 gm. of pamaquin three times daily for 3 days, followed by 0.1 gm. of quinacrine daily except Sunday until 60 days had elapsed from the initiation of treatment. The patients in Group III received the same prolonged treatment as did those in Group II, except that pamaquin was omitted, starting immediately after completion of the initial treatment with quinacrine.

†All relapses noted were proved by the finding of a positive blood smear with the exception of 2 patients, they occurred within 180 days after completion of treatment (Table 3). The mean days of observation of the patients without relapses and the probable errors of the means were as follows: Group I, 143.2± 7.4; Group II, 119.9± 3.7; Group III, 126.0± 4.6. The differences between the means were in all instances less than three times the probable errors of the differences.

‡Includes 4 patients who had relapses while receiving small doses of quinacrine (Table 4).

the first month after completion of suppressive treatment (compare Groups II and III with Group I) may be related to the fact that plasma quinacrine concentrations at the completion of treatment of

TABLE 3. *Length of Time before Relapses.**

TIME INTERVAL BEFORE RELAPSE†	GROUP I (124 CASES)		GROUP II (86 CASES)		GROUP III (77 CASES)		TOTAL (287 CASES)	
	RELAPSES		RELAPSES		RELAPSES		RELAPSES	
	No.	Percentage‡	No.	Percentage‡	No.	Percentage‡	No.	Percentage
days								
Less than 30	6	7	5	10	11§	23	22§	11
30–59	45	55	23	54	25	74	93	60
60–89	21	78	15	83	8	90	44	82
90–119	12	91	5	92	2	94	19	92
120–149	3	95	2	96	1	96	6	95
150–179	5	100	1	98	1	98	7	99
180–209			1	100	1	100	2	100
Totals	92		52		49		193	

*For explanation of treatment in Groups I, II and III, see footnote to Table 2.

†Calculated from time of completion of quinacrine treatment.

‡Cumulative.

§Includes 4 patients who had relapses while receiving prolonged treatment with small doses of quinacrine.

the patients in Group I averaged approximately 45 microgm. per liter, as compared with 25 microgm. in Groups II and III (Fig. 1).

Although the use of 0.1 gm. of quinacrine daily for sixty days had no significant effect on the ultimate relapse rate, there were only 4 relapses among 163 patients while they were on such continued small doses (Table 4). This extremely low incidence supports the observation in overseas theaters that the supervised administration of this dosage is an effective means of suppressing clinical malaria.[8]

In the two groups of patients given continued small doses, plasma quinacrine concentrations were determined each day during the initial week of treatment. As shown by Table 5, there was no relation between the plasma concentrations and the rate of relapse.

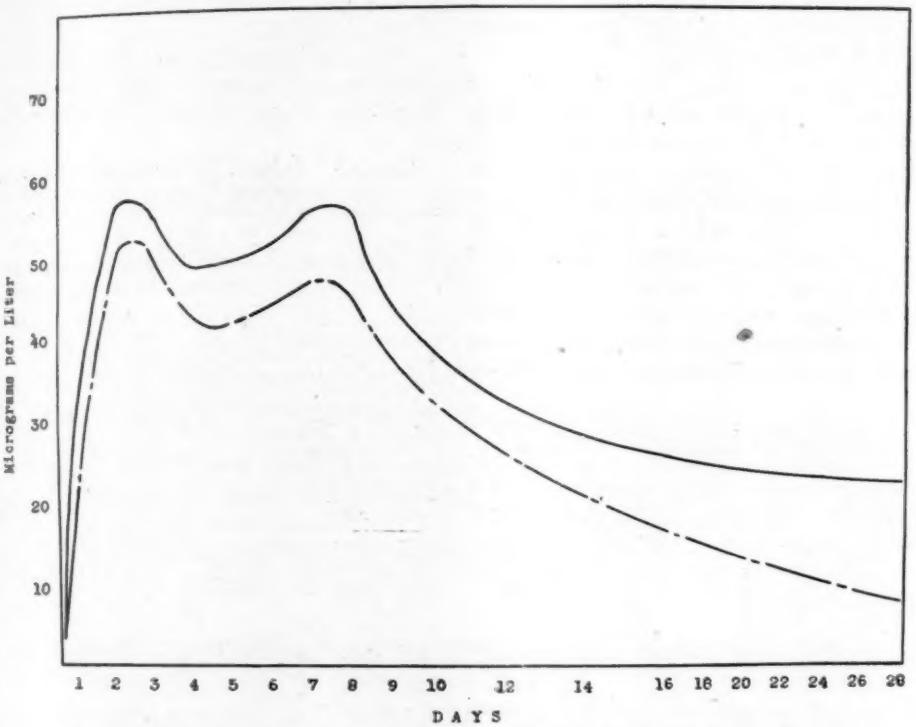

FIGURE 1. *Comparison of Plasma Quinacrine Concentrations at Different Times.*

The broken line represents the average fasting plasma level in 104 patients given 2.8 gm. of quinacrine in one week; the solid line that of 226 patients given the same amount of quinacrine in one week, followed by 0.1 gm. of the drug on six days of each week. The latter specimens were taken 4 hours after administration of 0.1 gm. of quinacrine.

LABORATORY FINDINGS

Plasma quinacrine concentrations. The plasma concentrations[9] obtained with the oral administration of 2.8 gm. of quinacrine in one week were determined in 226 patients during attacks. The results (Fig. 1) are comparable to those reported by Shannon and his co-workers.[10] The average fasting concentrations from the second through the eighth day were 42 to 51 microgm. per liter, whereas the average postprandial values two to four hours after breakfast — on the second through the seventh day quinacrine was given three times a day with meals — were somewhat higher, being 49 to 57 microgm. per liter. With these concentrations the symptoms of the attack were usually abolished within seventy-two to ninety-six hours. No attempt was made to determine the minimum effective dose, which presumably varied with the density of the parasitemia and the patient's immunity. Following cessation of quinacrine therapy,

TABLE 4. *Relapses during Treatment with 0.1-gm. Daily Doses of Quinacrine (4 Cases).**

CASE NO.	INTERVAL AFTER COMPLETION OF QUINACRINE THERAPY	PLASMA QUINACRINE LEVEL ON DAY OF RELAPSE	PRECIPITATING FACTORS
	days	*microgm./l.*	
1	20	24	None known
2	35	35	Measles
3	5	30	Lobar pneumonia
4	37	15	Repeatedly low plasma concentrations during four relapses

*These patients had smears and plasma quinacrine determinations every 5 days during the period of suppressive therapy. All of them had parasitemia, a temperature of over 100°F. and generalized aches, with the exception of one (Case 1), whose highest temperature reading was 99.2°F.

TABLE 5. *Relation of Relapse to Average Plasma Concentration of Quinacrine.**

PLASMA QUINACRINE LEVEL‡	GROUP II†		GROUP III†	
	NO. OF PATIENTS	PERCENTAGE OF RELAPSES	NO. OF PATIENTS	PERCENTAGE OF RELAPSES
microgm./l.				
20–39	27	41	17	59
40–59	35	63	37	65
60–79	24	79	20	65
80 or higher	0	0	3	66
Totals	86		77	
Average percentages		60		64

*No patient was counted who had not been observed either until a proved relapse had occurred or for 60 days following cessation of treatment.
†See footnote to Table 2 for outline of treatment in Groups II and III.
‡Average value during first week of treatment.

the plasma concentration slowly fell, so that by four weeks after the initial dose the average value was 8 microgm. per liter. The latter level is below the effective suppressive treatment level of 20 to 25 microgm. attained when 0.1 gm. of quinacrine was administered six days a week.

Complement-fixation reaction. In the past, attempts to obtain a specific complement-fixation reaction with serums from human beings infected by *P. vivax* have led to inconclusive results. Since there are no certain clinical criteria indicative of a complete cure in vivax malaria, a complement-fixation test that could detect latent malaria would obviously serve an extremely useful purpose. The results of such a test with the use of an antigen prepared by Coggeshall and Frisch[11] from the chicken parasite *P. gallinaceum* are shown in Table 6. There

TABLE 6. *Results of Complement-Fixation Tests during 234 Malarial Attacks and during Intervals Between 100 Observed Attacks.*

DAY OF ATTACK	NO. OF TESTS	NO. OF POSITIVE TESTS	NO. OF ANTI-COMPLEMENTARY TESTS	PERCENTAGE OF NEGATIVE TESTS
1st	234	123	11	43
2nd	234	130	8	41
3rd	234	109	18	46
4th	234	127	10	41
5th	234	121	13	43
Interval tests	891	287	30	64

was a maximum of 58 per cent positive tests on any one of five successive days during 234 recurrent attacks. During intervals between 100 proved attacks, 64 per cent of 891 tests made at five-day intervals were wholly negative. Although the complement-fixation test gave a group reaction for the serums of human beings infected by *P. vivax*, it did not detect latent malaria as proved by subsequent relapses in a sufficiently high percentage of cases to be of practical value.

Liver-function tests. Liver function was investigated in patients with recurrent attacks of malaria (Table 7). During the attacks transient disturbances were noted, as indicated by the results with the icteric index, serum bilirubin concentration, urine urobilinogen, bromsulfalein-clearance and cephalin-flocculation tests. Following attacks, only 5 per cent or less of the patients showed evidence of impairment by the cephalin-flocculation (Wilson antigen), bromsulfalein and galactose-tolerance tests. With a more sensitive reagent (Difco antigen), 20 per cent of the cephalin-flocculation tests were abnormal, but since neither antigen is standardized, this divergence in sensitivity cannot be interpreted. With both antigens, however, the trend was toward normalcy after the acute attack had subsided. Neither multiple attacks of malaria nor treatment with quinacrine resulted in evidence of permanent hepatic dysfunction.

SUMMARY AND CONCLUSIONS

Clinical and laboratory findings are presented for 808 soldiers admitted to the Malaria Section of the Harmon General Hospital from Pacific areas.

In 864 clinical attacks proved by positive smears *Plasmodium falciparum* was demonstrated in only 1, and then as part of a mixed infection with *P. vivax*.

The administration of adequate doses of quinacrine caused prompt disappearance of parasites from the blood stream. Without lessening the need for good mosquito control, this indicates that early diagnosis and treatment of relapses will help to prevent the spread of the infection in this country.

Symptoms of the acute attack were rapidly controlled by the administration of quinacrine in doses that yielded a fasting plasma concentration of 40 to 50 microgm. per liter.

Following such treatment, 67 per cent of 297 patients had proved relapses in a period of six or seven months. Approximately 50 per cent of these relapses occurred within sixty days after completion of treatment. There was no relation between the plasma quinacrine levels obtained and the rate of relapse.

The complement-fixation test with an antigen prepared from *P. gallinaceum* gave a positive reaction in approximately 50 per cent of tests performed on each of the first five days of 234 recurrences, but 64 per cent of 891 tests performed during the intervals between 100 proved attacks were wholly negative. In its present form the test does not detect latent malaria, as proved by subsequent relapses in a sufficiently high percentage of cases to be of practical value.

Multiple tests of liver function during and following attacks showed no evidence of permanent hepatic dysfunction.

TABLE 7. *Incidence of Abnormal Tests of Liver Function during and following Attacks of Malaria.*

TYPE OF TEST*	NO. IN GROUP		PERCENTAGE OF ABNORMAL TESTS					
	DURING ATTACK	AFTER ATTACK	DURING ATTACK					AFTER ATTACK
			Day of Illness					
			1	2	3	4	5	
Serum icteric index	108	260	16	12	2	1	1	3
Serum bilirubin	108	260	20	15	4		1	4
Urine urobilinogen	176	227	20	19	14	4	3	1
Bromsulfalein retention	151	149		4			1	3
Cephalin flocculation (Difco antigen)	42	204	71	86	88	81	86	20
Cephalin flocculation (Wilson antigen)	42	204	10	29	21	17	29	5
Galactose tolerance		207						3
Serum protein		172						2
Serum cholesterol		156						0
Serum phosphatase		191						0

*The following standards were used as an index of abnormality: icteric index, more than 8 units per 100 cc.; serum bilirubin, more than 0.5 mg. per 100 cc.; urine urobilinogen, positive in a dilution higher than 1:2; bromsulfalein, retention of more than 4 per cent in a specimen of blood taken 45 minutes after injection of 5 mg. per kilogram; cephalin flocculation, positive flocculation; galactose tolerance, excretion of more than 3 gm. in 5 hours after ingestion of 40 gm. of galactose; serum protein, less than 6.0 gm. per 100 cc.; serum cholesterol, more or less than 150-250 mg. per 100 cc.; serum phosphatase, more than 5 Bodansky units per 100 cc.

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LONGEVITY WITH VENTRICULAR ANEURYSM

Report of a Case with a Survival Period of Fifteen Years

CAPTAIN SIDNEY L. PENNER, M.C., A.U.S., AND CAPTAIN MICHAEL PETERS, M.C., A.U.S.

THE duration of life after an acute coronary-artery occlusion has long been regarded as brief, and it is only in recent years that the more optimistic viewpoint pronounced in 1912 by Herrick[1] has been justified. In increasing numbers, cases have been reported of survivals for as long as seventeen, twenty-four, and even forty years.[2–5] not so much because of the presence of the outpouching of the ventricular wall per se, as because this phenomenon is indicative of extensive myocardial damage. Cases have been reported, however, in which the patients have survived for as long as eight to ten years.[9, 10] We present a case in which the patient survived a severe attack of myocardial infarction with subsequent ventricular aneurysm and was alive and in reasonably good health fifteen years after the acute episode.

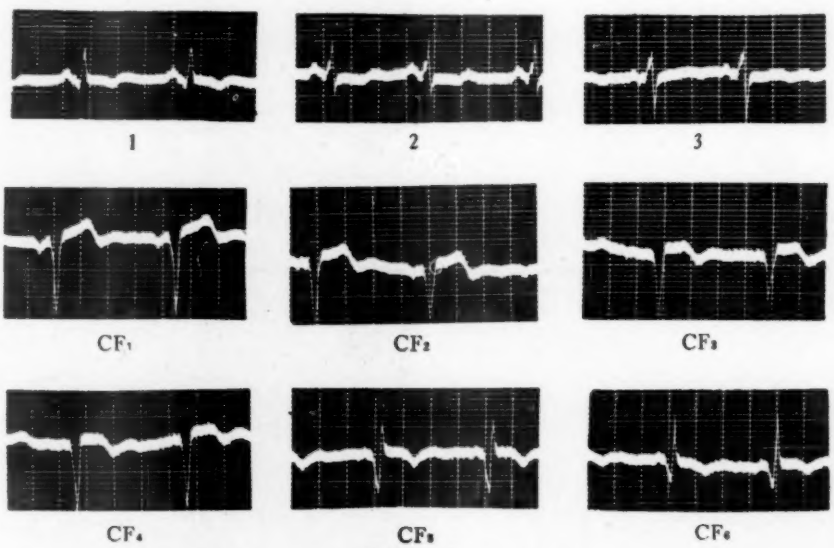

FIGURE 1.

As an explanation of this change in prognosis, it has been suggested[6] that the myocardial infarction that is being diagnosed in mild and atypical forms would have been dismissed years ago as so-called "acute indigestion" or never have been brought to the attention of a physician, and that these forms have a more favorable prognosis than do the severer ones.

The prognosis of cases of ventricular aneurysm has heretofore been considered as particularly grave,[6–8] and the diagnosis has long been regarded as one to be made only at autopsy.[7] This is true

CASE REPORT

A 43-year-old infantry officer on October 25, 1929, had an episode of severe pain in the precordium and left arm while crossing a parade ground. He was unable to walk farther because of the pain, which gradually subsided when he stood still. He had no further symptoms until November 18, when while reading he was seized with agonizing chest pain, radiating to the left arm. He was extremely weak and restless but was not dyspneic. The examining physician found the patient in a state of shock, with mild cyanosis, profuse sweating, a cold clammy skin and a barely perceptible pulse.

Severe pain persisted for several hours despite the administration of 73 mg. of morphine. During the following several days the temperature was elevated, reaching a maximum of 102°F. A loud pericardial friction rub was heard, and rales were audible at the base of the left lung. The blood pressure was 120/76. On December 3, the patient was seen in consultation by Dr. George H. Anderson, of Spokane, Washington. Inverted T waves were noted in the electrocardiogram. No further details of this examination are available.

For the first 6 weeks after the onset of the illness, the patient remained in bed at home and his general condition steadily improved. On January 31, 1930, he had a recurrence of pain, less severe than before but again followed by an audible pericardial friction rub. In February, he was transferred to a general hospital, where examination showed a traumatic cataract of the right eye and moderate thickening of the radial and brachial arteries. The blood pressure was 128/84. The heart was not enlarged, but the sounds were of poor quality and a soft systolic murmur was heard at the apex. There were no signs of congestive failure. The electrocardiogram showed a typical coronary T wave and ST interval in Lead 1. On March 30, there was another attack of severe cardiac pain, followed by slight fever for 4 or 5 days. The patient gradually improved from this time on but remained in the hospital until February, 1931, when he was retired from the Army.

For the next 2 years he had slight pain in the right chest and left shoulder and arm but no other symptoms. After 1934 he felt well and was active in building and sailing boats and doing deep-sea fishing. In September, 1942, because of the war, he was returned to limited duty and was able to work in an administrative capacity without symptoms until September, 1944. At that time he began to complain of pain in the left shoulder, radiating down the left arm and hand. There was occasional substernal distress, the pain being present at rest as well as on exertion. Cramps developed in the calves on walking and also on occasion during sleep. There was mild dyspnea on effort. An electrocardiogram was similar to those subsequently taken in April, 1945 (Fig. 1). Examination in that month showed thickening of the peripheral arteries. The blood pressure was 140/90. The heart was enlarged to the left on percussion, but there were no

FIGURE 2.

thrills or abnormal pulsations over the precordium.[11] The heart sounds were of good quality, and no murmurs were present. The first sound at the apex was split. No signs of congestive failure were present.

Electrocardiograms showed a regular sinus rhythm, left-axis deviation, slight slurring of the QRS complex in the limb leads, an inverted T wave in Leads 1, CF_3, CF_4, CF_5 and CF_6, elevation of the ST segment in Lead 1 and depression

knob. The roentgenologic interpretation was left-ventricular aneurysm with calcification of the outer ventricular wall and apparent cardiac enlargement because of rotatory displacement of the heart, due to ventricular aneurysm.

The laboratory findings, including a complete blood-cell count, urinalysis, determination of the blood sugar and urea and Kahn and sedimentation-rate tests, were normal. The patient was kept in the hospital for several weeks for study,

FIGURE 3.

of ST segments in Leads 2 and 3. The QRS complex in Leads CF_1, CF_2, CF_3 and CF_4 was completely inverted, and a large Q wave was present in Leads CF_5 and CF_6. X-ray examination of the heart showed the cardiac silhouette to be slightly enlarged (Fig. 2). Overlying the left ventricular portion of the cardiac silhouette was seen a curvilinear, thin band of calcification forming the outer half of a soft-tissue consensation. Left lateral films showed encroachment of the left atrium on the esophagus. Fluoroscopic examination indicated that the calcification was in the outer surface of an outpouching of the anterior surface of the left ventricle. No pulsation was noted in this area. Bucky films taken in the right anterior oblique position (Fig. 3) confirmed these findings and also showed calcification in the wall of the aortic

and on September 6, 1945, he was feeling well and again enjoying deep-sea fishing.

DISCUSSION

The diagnosis of cardiac aneurysm was not suggested by the physical findings,[11, 12] nor did the electrocardiographic findings fall into the pattern said to characterize this lesion,[13] although it was characteristic of extensive anterior myocardial infarction. The x-ray examination, however, made the diagnosis clear. The following roentgenographic

criteria have been suggested[8] for the diagnosis of ventricular aneurysm: enlargement of the left ventricle with deformity of the cardiac contour, a localized protuberance of the silhouette, absent or abnormal pulsations under the fluoroscope, evidence of adhesions to the chest wall and calcification of the aneurysmal wall or intraventricular clot. Of these criteria, the present case showed the first, second, third and fifth. In addition, there was dorsal displacement of the esophagus.[14, 15]

Although the diagnosis of aneurysm was not established until 1945, it is justifiable to assume that the aneurysm was a direct result of the infarction or infarctions of 1929 and 1930. Ventricular aneurysm has been found to develop during the acute stage.[16] The severity of the recurrent seizures, with pericarditis and fever as manifestations, and the persistence of symptoms requiring hospitalization for more than one year testify to the extent of the myomalacia. The present aneurysm had probably existed for the entire fifteen-year period and may have increased in size with the passage of time. The calcification must have been many years in development. The recurrence of symptoms in 1944 may have been due to one or more minor episodes of infarction, but the aneurysm was obviously of much earlier origin.

Since ventricular aneurysm has been found to develop even during the acute stages of myocardial infarction,[16] and since the time for healing of an infarcted area has occasionally been found to be prolonged,[17] the precaution of confining the patient to the hospital for over a year seems justified. During the next fourteen years, the patient reaped the benefit of his long convalescence, being able to lead an active and almost symptom-free existence. In this period of hospitalization, during which the demands on the myocardium were kept at a minimum, the formation of new anastomoses between the right and left coronary arteries and dilatation of the pre-existing anastomoses could take place.[18] The enlargement of the defect in the ventricular wall under the impact of systole was diminished,[19] and the danger of ventricular rupture was mini-mized.[20, 21] Had a less conservative approach[22-24] been employed, the result might have been disas-trous.

Summary

A case of ventricular aneurysm with a survival period of fifteen years is reported and is discussed in relation to the therapeutic implications.

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OSSEOUS GAUCHER'S DISEASE WITH MACROCYTIC NORMOCHROMIC ANEMIA

Report of a Case

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In the typical cases of Gaucher's disease, the disturbance of the cellular metabolism, characterized by the accumulation and retention of the cerebroside kerasin in the reticular and histiocytic cells, leads to the enlargement of the spleen and liver, with occasional osseous defects. In addition, there are pigmentation of the skin, pingueculae, leukopenia, microcytic, hypochromic anemia and hemorrhagic tendencies due to thrombocytopenia. In the case reported below, the most prominent manifestations of the typical case of Gaucher's disease were absent. In fact, the presenting finding was an anemia that was discovered by the patient himself while he was practicing routine red-cell counts using his own blood. Subsequent examination disclosed the unusual combination of a macrocytic normochromic anemia with Gaucher's disease in which the spleen and liver were not palpable. The diagnosis was made possible only by the examination of the sternal bone marrow, since the radiologic appearance of the bones was normal.

Case Report

A 20-year-old, Jewish laboratory technician was inducted into the Army in April, 1943. In the following summer, while undergoing basic training, he suffered pain in the right hip. This subsided in 4 days without treatment. Early in May, 1944, while the patient was playing baseball, another attack of pain developed in the right hip and lower part of the back, although there had been no trauma or extreme physical exertion. The patient was then practicing red-cell counts, using his own blood, and discovered that he had an anemia. Following this finding, a diagnosis of macrocytic anemia was made. Free hydrochloric acid was found in the stomach. In the meantime, the pain in the back, which had persisted, suggested the presence of a ruptured intervertebral disk, but examination of the cerebrospinal fluid on June 2 disclosed nothing of significance, and a neurologic examination was negative. The patient was given 15 mg. of thiamine daily, and the hip and back pain subsided.

On August 1, the patient reported to sick call complaining of epigastric distress, nausea and anorexia. Physical examination revealed a well developed and well nourished man not acutely ill. There was a slight yellowish tint in the scleras, as well as a malar flush and a generalized slight yellowish tint of the skin. The abdomen was soft. Tenderness was present in the right upper quadrant. The spleen was not palpable, and the edge of the liver, which was tender, could be felt only on deep inspiration. During his hospital stay, the patient complained of "gas on the stomach" and appeared sluggish and weak. The possibility of an attack of infectious hepatitis was entertained on account of the symptoms, the yellow tint in the scleras and skin, the tender liver and the increased blood bilirubin. Pain was still felt in the right hip. The feces were always formed and brown. The patient was treated with a high-carbohydrate, high-vitamin diet and bed rest. On August 21, he was returned to duty. It was planned to perform a sternal puncture in a month to elucidate the nature of the anemia.

On September 28, a sternal puncture was done and a diagnosis of Gaucher's disease of the osseous type was made. On October 8, the patient was readmitted for further study. He had been feeling sluggish and weak and had had a poor appetite. Pain had continued in the right hip and lower back. No other member of the patient's family suffered from Gaucher's disease, although an aunt died of an anemia the exact nature of which was unknown to the patient.

On physical examination, the spleen and liver were not palpable. The malar flush and the generalized slight yellow tint of the skin were still present. The yellowish tint in the scleras was discovered to be caused by definite bilateral pingueculae extending from the medial limbi of the corneae to the internal canthi. Roentgenograms of the entire skeleton revealed no lesions in the bones. On October 11, biopsies of the sternum and the left tibia were done. The sternal marrow did not look remarkable, but the tibia appeared devoid of marrow and a large amount of fluid blood came from the marrow cavity.

Pertinent data concerning the red cells and the hematologic indices and corpuscular constants are given in Table 1. On May 27, the white-cell count was 4850, with 45 per cent segmented neutrophils, 6 per cent band neutrophils, 43 per cent lymphocytes, 4 per cent monocytes and 2 per cent eosinophils. Slight anisocytosis and slight poikilocytosis were present. On August 17, the white-cell count was 7000, with 40 per cent segmented neutrophils, 4 per cent band neutrophils, 46 per cent lymphocytes, 7 per cent monocytes and 3 per cent eosinophils. On September 5, the white-cell count was 6900, with 46 per cent segmented neutrophils, 2 per cent band neutrophils, 38 per cent lymphocytes, 3 per cent monocytes and 1 per cent eosinophils. Slight anisocytosis and slight poikilocytosis were present. On September 25 it was 8600, with 42 per cent segmented neutrophils, 15 per cent band neutrophils, 35 per cent lymphocytes, 3 per cent monocytes, 1 per cent eosinophils and 4 per cent basophils. Slight poikilocytosis was noted. On October 8, it was 5300, with 42 per cent segmented neutrophils, 12 per cent band neutrophils, 41 per cent lymphocytes, 3 per cent monocytes and 1 per cent eosinophils. Polychromatophilia, achromasia,

Table 1. *Summary of Pertinent Laboratory Data.*

Date	Hemoglobin (Sahli)	Red-Cell Count	Hematocrit	Reticulocytes	Volume Index*	Color Index*	Mean Corpuscular Volume	Mean Corpuscular Hemoglobin	Mean Corpuscular Hemoglobin Concentration
	gm./100 cc.	*x10⁶*	%	%			*cu. microns*	*micromicrogm.*	%
5/27	—	4.1	—	0.9	—	—	—	—	—
7/15	12.5	4.0	—	—	—	—	—	—	—
8/17	13.0	3.6	40		1.27	1.26	112	36.4	32.5
9/5	13.0	3.7	43	0.9	1.35	1.23	117.8	35.6	30.2
9/25	13.4	3.7	40	0.6	1.25	1.26	108.9	36.5	33.5
10/8	13.5	3.3	44	1.0	1.53	1.41	133.3	40.9	30.6
Normal					1.00	1.00	82–92	27–31	32–36

*The figure of 43.2 per cent was taken as the normal mean volume of packed red cells, and 14.5 gm. per 100 cc. as the normal hemoglobin.

anisocytosis and poikilocytosis were present. No spherocytes were ever found. The platelet count was 250,000.

A red-cell fragility test on August 5 revealed that hemolysis began at 0.46 per cent saline solution for the patient and 0.46 per cent for the control and was complete at 0.36 per cent for the patient and 0.38 per cent for the control. Another test on October 8 showed that hemolysis began at 0.44 per cent for the patient and 0.46 per cent for the control and was complete at 0.36 per cent for the patient and 0.38 per cent for the control. The sedimentation rate (Westergren) was 11 mm. per hour on August 6 and 24 mm. on August 19, 1944. Two urinalyses were negative. Three Wallace–Diamond tests for urine urobilinogen disclosed no increase over the normal.

On May 26, 1944, a gastric analysis revealed 12 to 94 units of free hydrochloric acid and 28 to 106 units of total acidity. On May 28, no excess fat was found in the feces. On August 8, the blood nonprotein nitrogen was 33.9 mg. per 100 cc. and the blood cholesterol 200 mg.

The icteric indices and the corresponding quantitative bilirubin determinations (Van den Bergh reactions) are listed in Table 2. The Van den Bergh reaction was always indirect.

On August 7, the total serum protein was 8.2 gm. per 100 cc. and the albumin-globulin ratio 2:1. On August 8, the total serum protein was 7.2 gm. and the albumin-globulin ratio again 2:1. In the Quick hippuric acid liver-function test (oral), the total excretion of hippuric acid amounted to 5.5 gm. On June 2, there were no white cells in the spinal fluid and the total protein was 32.3 mg. per 100 cc.

TABLE 2. *Icteric Indices and Blood Bilirubin Levels.*

DATE	ICTERIC INDEX	BLOOD BILIRUBIN *mg./100 cc.*
5/9	15.5	—
6/11	10.0	—
8/1	10.0	2.60
8/4	10.0	2.30
8/12	9.0	2.60
8/19	9.0	1.90
8/28	7.0	0.25
9/5	12.0	2.30
9/25	11.0	1.50

The sternal-puncture material obtained on September 28 was smeared, and the small amount of blood clot remaining, which amounted to 0.4 cc., was placed in Zenker's fluid for sectioning. Staining of the direct smear with Wright's stain revealed 5 per cent segmented neutrophils, 32 per cent band neutrophils, 14 per cent juvenile neutrophils, 12 per cent neutrophilic myelocytes, 2 per cent myeloblasts, 2 per cent pronormoblasts, 11 per cent normoblasts, 18 per cent lymphocytes, 2 per cent monocytes, 1 per cent eosinophils and 1 per cent mitotic figures. In a general survey of the bone-marrow smears, scattered large cells were found, especially at the edges. These cells possessed abundant cytoplasm staining a light blue. Within the cytoplasm there were scattered striations, giving it a reticulated appearance. Infrequently a small vacuole was seen, and more rarely a purplish dot was found near the nucleus. The nuclei varied in shape, some being round and others slightly indented. In addition, multilobated and multinucleated types were noted. The chromatin pattern formed a coarse, open meshwork. The small blood clot fixed in Zenker's fluid was handled by the usual method for tissues. Examination of paraffin sections stained with hematoxylin and eosin revealed the presence of small fragments of marrow within the blood clot. Much of the marrow had been extensively replaced by compact masses of Gaucher cells. These large cells possessed abundant delicately striated cytoplasm. Many of the striations were arranged in concentric lines about the nucleus. Frequently the cytoplasm was diffusely yellowish brown. Ingestion of red cells by occasional Gaucher cells was noted, especially by those the cytoplasm of which was pigmented. Infrequently, acicular spaces were noted in the cytoplasm. Many of the nuclei were multiple or multilobated, whereas others were round or indented. Most of the nuclei were vesicular, with occasional large nucleoli. No mitotic figures were seen among these large cells. Among the marrow cells still remaining there was active hematopoiesis, with normoblasts predominating. An occasional mitotic figure was found among the marrow cells. With the Giemsa stain, the Gaucher cells were pale, the cytoplasm being devoid of color. Otherwise the picture was similar to that found in the sections stained with hematoxylin and eosin. The Gaucher cells in sections stained with Mallory's aniline blue-orange G method were extremely prominent because their cytoplasm was stained blue. The cytoplasmic striations appeared to be accentuated, giving the cytoplasm a wrinkled appearance, and occasionally large vacuoles appeared in it.

The sternal marrow obtained by biopsy on October 1 was treated in the same way as was the sternal-puncture material. A direct smear stained with Wright's stain contained 8 per cent segmented neutrophils, 22 per cent band neutrophils, 8 per cent juvenile neutrophils, 13 per cent neutrophilic myelocytes, 4 per cent myeloblasts, 4 per cent pronormoblasts, 14 per cent normoblasts, 13 per cent lymphocytes, 10 per cent Gaucher cells, 3 per cent monocytes and 1 per cent mitotic figures. Megakaryocytes and platelets were few. The appearance of the cells was similar to that in the direct smear of the sternal puncture. The marrow in the tissue fixed in Zenker's fluid was similar to that found in the blood clot obtained by the first sternal puncture, except that the material was more abundant. In addition, the fatty tissue in the marrow was scanty and some of the Gaucher cells assumed a spindle appearance. The bone trabeculae were not remarkable. The Gömöri stain for hemosiderin stained the yellowish-brown pigment in the cytoplasm of the large cells blue, and the pigment was thus identified as hemosiderin. Sudan IV, the Schultz stain for cholesterol and the Smith–Dietrich stain were applied to direct smears of the sternal marrow, but none disclosed the presence of lipoids. The biopsy of the tibial marrow yielded little material. The direct smear consisted mainly of red cells, with a rare Gaucher cell. In paraffin sections, a small fragment of marrow was found in a blood clot that possessed the characteristics of the sternal marrow. A diagnosis of Gaucher's disease was made after microscopical examination of both the sternal and tibial marrows. Biopsy examination of the skin of the right flank on October 23 revealed no pigmentation or other abnormal details.

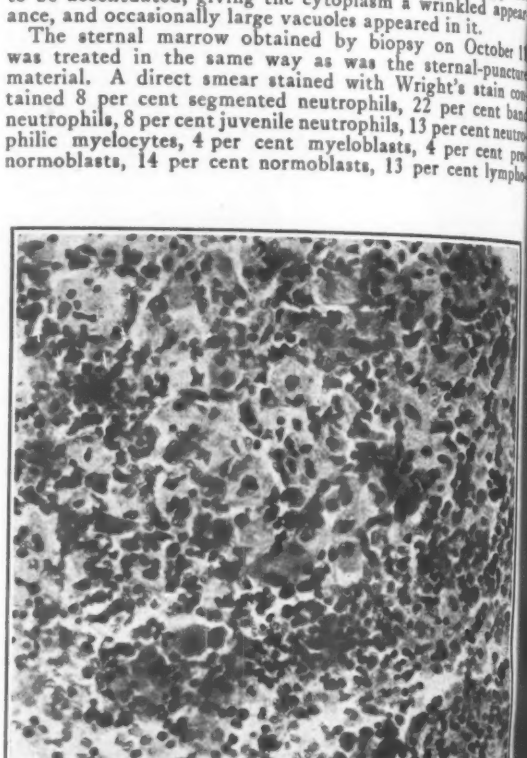

FIGURE 1. *Group of Gaucher Cells Infiltrating the Sternal Marrow (x 225).*

DISCUSSION

Several characteristics of this case deserve comment. Outstanding is the macrocytic, normo-

chromic anemia, a normochromic anemia being defined as one with a mean corpuscular hemoglobin concentration of 30 per cent or more. The lack of a palpable spleen and liver and of roentgenologic changes in the skeleton and the presence of bone pain and latent jaundice also merit attention.

According to Thannhauser,[1] the anemia in Gaucher's disease is usually microcytic, although he admits that the anemia may become macrocytic in the terminal stages of the disease. Pick[2] and Capper, Epstein and Schless[3] state that the anemia is usually hypochromic, whereas Wintrobe[4] describes it as normocytic and Mandlebaum and Downey[5] call it chlorotic. Bloem, Groen and Postma[6] reported 4 cases. In 1 the mean corpuscular volume was 108 cubic microns with a color index of 1.2, but the other 3 showed a hypochromic anemia. In the case reported by Melamed and Chester,[7] there was a marked hypochromic microcytic anemia, but one examination out of twenty-one showed a macrocytosis. Finally, there was a hypochromic anemia in Petit and Schleicher's[8] case. It is thus evident that the finding of a macrocytic normochromic anemia is unusual, but in the light of Davidson and Fullerton's[9] report on the rare types of macrocytic anemia it is not unexpected, since infiltration of the marrow by the neoplastic cells found in some of their cases closely resembles the mechanism present in osseous Gaucher's disease, although the Gaucher cells are not per se neoplastic.

Most other writers are agreed that enlargement of the liver is usually an accompaniment of an enlarged spleen. As for the spleen, Thannhauser[1] states that it is always enlarged, and Krumbhaar[10] emphasizes the importance of the size of the spleen in making a diagnosis of Gaucher's disease. Pick,[2] on the other hand, after studying cases of bone involvement by Gaucher cells in which the weight of the spleen and liver was less than the average weight of these organs in Gaucher's disease, directed attention to an osseous form that he considers to belong to a subgroup having a special constitutional predisposition for the skeletal system, so that occasionally all members in a family afflicted with the disease have a predominant involvement of the bones. Thannhauser[1] prefers not to set up a separate category but holds that the involvement of the skeleton is as constant as is involvement of the spleen. The latter is likely, but why the spleen should remain small in cases of Gaucher's disease in which the skeleton is involved, as in our case, is puzzling; nor is the solution of the puzzle advanced by the disclosure of the presence of Gaucher cells postmortem in a spleen weighing 100 gm. in the case of Petit and Schleicher,[8] in which there was also marked bone involvement. In the present case, the explanation may lie in the early stage of the disease, but this seems less probable in the case of Petit and Schleicher,[8] in which the patient was seventy-nine years old.

It must be emphasized that the bones may be infiltrated by Gaucher cells without visible changes in the roentgenograms. This phenomenon is evident in our case and in that of Petit and Schleicher,[8] and in both, diagnosis was made possible only by the examination of the sternal marrow. The diagnosis of Gaucher's disease in a man of fifty-two reported by Vogel, Erf and Rosenthal,[11] in whom no enlargement of the spleen or liver could be found, was also made by sternal puncture. In this case rarefaction of the bones could be seen in the roentgenogram.

In many cases of osseous Gaucher's disease, the presence of bone pain has led to erroneous diagnoses. Thannhauser[1] points out that bone pain, accompanied by fever, may be an early symptom and may simulate rheumatic pain. On the other hand, acute osteomyelitis may be mistakenly diagnosed, as happened in the case of Capper, Epstein and Schless.[3] Welt, Rosenthal and Oppenheimer[12] also emphasize the presence of bone pain in this disease. In the present case, the diagnosis of a ruptured intervertebral disk was considered on account of the low-back pain, but a negative neurologic examination and normal findings in the cerebrospinal fluid aided in discarding this possibility.

The occurrence of jaundice is considered by Krumbhaar[10] to be rare, whereas Thannhauser[1] states that a slight transient jaundice is an exception. According to Mandlebaum and Downey,[5] it is absent. The last authors refer to jaundice that is clinically perceptible. A perusal of the reports in the literature, however, discloses the presence of latent jaundice in some cases of Gaucher's disease, including the osseous types. In fact, it was present in one of Thannhauser's[1] cases, and the statement that there may be a positive indirect van den Bergh reaction in cases of Gaucher's disease may be found in his review. In addition, latent jaundice was found in the case of Petit and Schleicher.[8]

The explanation of the mechanism of the production of latent jaundice in the osseous cases without enlargement of the liver has not been found in the literature and is open to speculation, especially since in the autopsied case of Petit and Schleicher[8] no Gaucher cells were found in the liver, which appeared little altered from the normal. In the latter case, a hemolytic process may have been active, although no evidence was presented to support this. A definite hemolytic process was present, however, in a case of Gaucher's disease reported by Mandelbaum Berger and Lederer,[13] in which a diagnosis of hemolytic anemia was made. The possibility exists that a hemolytic process was active in our case also, but unfortunately no elevated urinary urobilinogen excretion was revealed by the Wallace–Diamond test, nor could reticulocytosis be detected. On the other hand, the presence of abundant hemosiderin in the Gaucher cells and the active erythrophagia by the same cells in the marrow in the present case

suggest that destruction of red cells was proceeding, but at such a slow rate that reticulocytosis and increased urinary urobilinogen did not appear. It was impossible to determine the amount of fecal urobilinogen excreted.

The deposition of hemosiderin in Gaucher's disease has been emphasized by Pick,[2] who believes that there is a storage of hemoglobinogenic pigments in lipoid-storing cells. In fact, he states that hemosiderosis — or rather, increased blood destruction — occurs when the reticuloendothelial system is stimulated. This theory is probably too general, since hemosiderosis does not occur in Niemann–Pick's disease,[2] although here other factors, such as the age of the patient and the rapidity of the course of the disease, may contribute to the absence of the hemosiderin. It is well established that hemosiderin is either absent or extremely scarce in children with Gaucher's disease.[2] It is thus possible that erythrophagia and hemosiderosis vary in intensity in cases of Gaucher's disease and that it is in those in which the hemolytic process is at least moderately active that latent jaundice or even frank jaundice occurs.

That the episode of epigastic distress, nausea and anorexia in the case reported herein was due to an attack of infectious hepatitis is doubtful. Certainly at that time there was no rise in the blood bilirubin over that usually found in this patient, and there was no increase in the size of the liver.

It is true that the presence of the Gaucher cells in the marrow, with their characteristic wrinkled cytoplasm staining blue with Mallory's aniline blue-orange G stain and containing hemosiderin but no stainable lipoid, made possible an unequivocal diagnosis, but before an examination of the marrow could be carried out other diseases in which a macrocytic normochromic anemia and an elevated blood bilirubin occur had to be ruled out.

Some of the diagnostic possibilities were pernicious anemia, nutritional macrocytic anemia, achrestic anemia, cirrhosis, congenital and acquired hemolytic jaundice and familial nonhemolytic jaundice. Sickle-cell anemia may be macrocytic and is accompanied by a hyperbilirubinemia, but this was not seriously considered on account of the race of the patient. Most of these possibilities were discarded through the data obtained from the clinical history, the physical examination and the laboratory studies other than that of the sternal marrow. Of all the suggested conditions, achrestic anemia and familial nonhemolytic jaundice remained after the above studies were carried out. These were quickly discarded after an examination of the sternal marrow. Certainly, the demonstration of Gaucher's cells in the sternal marrow in this case offers another example of the value of sternal biopsy or puncture in the elucidation of blood dyscrasias.

Summary

The diagnosis of a reported case of Gaucher's disease was made difficult by the presence of a macrocytic normochromic anemia with latent jaundice and by the lack of an enlarged spleen and liver, together with the lack of roentgenologic changes in the bones.

The sternal puncture alone made possible the diagnosis by the demonstration of Gaucher cells in the marrow. Once again, the value of sternal puncture in differentiating the blood dyscrasias has been proved.

It is suggested that the probable mechanism responsible for the production of the latent jaundice rested on the active erythrophagia of the Gaucher cells.

We are indebted to Dr. L. C. D. Hermitte, pathologist of the Sheffield Royal Infirmary, for the suggestions concerning the pathogenesis of the latent jaundice and for the photomicrograph.

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MEDICAL PROGRESS

SURGERY OF THE HEART AND STRUCTURES RELATED TO IT (Concluded)

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BOSTON

TRAUMA

Wounds

What can be accomplished in the reduction of the mortality from wounds of the heart is ably demonstrated in a paper by Elkin[13] from the Department of Surgery at Emory University. Abstracts of the histories of 23 patients operated on for wounds of the heart are presented. In this series, the mortality rate was 22 per cent, as compared with 42 per cent in 38 cases previously reported from the same department. Although it has generally been considered that the intravenous administration of fluids prior to operation in cases of acute pericardial tamponade is contraindicated because of the danger of increasing the hemorrhage and hence the degree of tamponade, Elkin believes that such infusions have a beneficial effect by increasing the blood volume and hence the cardiac output. This point of view is based on the experimental work of Cooper et al.,[14] who produced pericardial tamponade in dogs and then gave saline solution intravenously, with striking improvement.

Immediate operation was undertaken in all Elkin's cases, being performed as soon as the diagnosis had been made. As evidenced by the reported cases from other clinics, however, aspiration of the pericardium, either as a temporary or a definitive measure for the treatment of cardiac tamponade, may be properly employed.

Foreign Bodies

During the course of a war characterized by a high caliber of the medical services, many noteworthy papers dealing with the surgery of trauma of the thorax have naturally appeared. The experiences of Harken and his associates,[15–17] in connection with foreign bodies in and close to the thoracic blood vessels and heart are particularly worthy of review, since they deal with practical aspects of intracardiac surgery, hitherto largely of theoretical conception as regards human beings.

These workers have removed missiles from within or adjacent to the great vessels of the thorax in 79 cases, in 3 of which the missiles were embolic. The latter were removed by Harken.[18] His search of the literature revealed nearly 40 cases of embolic foreign bodies, but there were no examples of successful removal of embolic missiles from the great intrathoracic blood vessels. Harken assumes, however, that when all the reports of thoracic surgery performed in World War II have been published, these will include additional cases of successful removal of embolic missiles from these vessels. In the first of Harken's cases with removal, adequate roentgenograms were taken before embolization, and the embolus apparently moved from the liver to the left pulmonary artery. In the second case, movement was apparently through the heart and the embolus lodged in the innominate artery. In the third case, the embolus moved at operation from the left to the right pulmonary artery and was successfully removed from the right side at a second operation.

Harken and his associates state that certain foreign bodies impinging on the great vessels of the chest are removed to prevent erosion and hemorrhage or infection. In 15 per cent of the cases in this series the missiles were in abscesses; in 30 per cent, they were associated with other foreign material, such as bone or clothing; and in 67 per cent, they grew pathogenic organisms on culture.

In 59 cases, foreign bodies were removed from within or in immediate relation to the heart. In 13 of these, they were taken from the chambers of the heart. The indications for the removal of foreign bodies in the heart were originally set up on the basis of the medical literature, Harken's experimental work on dogs before the war and certain theoretical grounds. They are as follows: to prevent embolus (a foreign body or thrombus); to reduce the danger of bacterial endocarditis; to prevent recurrent pericardial effusions; and to diminish the incidence of cardiac rupture or myocardial hernia. It was believed that all four of these indications were supported by their own clinical experience and that of their colleagues. In addition, pain and cardiac neurosis may constitute indications for intervention.

Harken emphasizes that he and his associates elected to leave behind more foreign bodies than they elected to remove. Size and clinical manifestations were inevitable factors influencing their decision to operate. Small foreign bodies were thought possibly to be associated with less damage and to be less likely to cause the above complications than larger ones. They were, moreover, technically more difficult to remove. Inasmuch as it cannot be definitely ascertained from the literature just how hazardous retained intracardiac

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foreign bodies are, and because some patients have undoubtedly remained asymptomatic for many years, the authors are reluctant to be dogmatic. They believe, however, that figures in the literature are misleading, because in many cases only roentgenograms were used in localizing the foreign body that was called intracardiac. They doubt whether some of these missiles were intracardiac, since in their own experience, in less than half the cases referred to them as having intracardiac foreign bodies, these were found on careful fluoroscopic examination to be in the heart. Furthermore, almost a third of the missiles that had been regarded as intracardiac were found at operation to be extracardiac. As these authors' experience widened, they felt justified in broadening their indications, since they had no deaths and since certain complications, some of them lethal, were observed to occur when the foreign bodies were left alone.

In the surgical procedure followed by Harken et al., the approach to the heart varies with the individual case and is planned so that minimal dislocation of the heart is necessary. The sternum, cartilage and ribs may be divided, but nothing is removed and no tissue is discarded. When adequate exposure is obtained, two rows of U sutures are placed on either side of the proposed cardiotomy incision. These sutures are used as hemostatic sutures between manipulations and after the foreign body has been extracted with forceps. The myocardium is closed by approximating the margins of the incision with the inner row of hemostatic sutures and using the outer row to hold a pericardial graft in place.

In a dramatic case, three successive cardiotomies were performed at suitable intervals before the foreign body, which migrated twice, was successfully removed. It was originally in the right ventricle but moved to the right auricle at the first operation. When approached from the right side at the second operation, it moved from the right auricle back to the right ventricle. Because the patient was placed in the reverse Trendelenburg position with the right side elevated, the foreign body remained in the right ventricle during the third cardiotomy, when it was successfully removed. Reoperation in this case gave a valuable opportunity to note the manner of healing of the myocardial incision. The myocardial scar was firm and well healed six months after the original incision, a fact that to Harken offers assurance that the injury to the myocardium caused by the incision and exploration of the ventricle is not significant.

The cases previously referred to and subsequent ones represent 138 patients and one hundred and forty-three operations. There were no deaths.

Acute Pericarditis

Streptococcus viridans is rarely encountered as the causative agent in purulent pericarditis. Hargrove[19] reports a case in a sixteen-year-old boy with acute pericarditis resulting from a *Str. viridans* septicemia that followed an upper respiratory infection. The organism was twice found in cultures of blood and pericardial fluid. The author suggests that electrocardiograms be made during septicemia, pneumonia and osteomyelitis if there is a suspicion of pericardial disorder. In the case reported, pericardicentesis and electrocardiography indicated acute pericarditis. The patient was treated by aspiration and sulfanilamide, but the disease ran a stormy course and the outcome was at first doubtful, although there was an eventual recovery.

Wise and Shafer[20] report a case of pericardial effusion secondary to lobar pneumonia in a woman of thirty-one. Sulfadiazine and intramuscular penicillin had no effect on the lesion, but there was dramatic response to the intrapericardial injection of penicillin following pericardicentesis and the removal of 70 cc. of turbid blood-tinged fluid. A dose of 40,000 units of penicillin in 30 cc. of normal saline solution was introduced directly into the pericardial cavity. Within sixteen hours the temperature dropped to normal. Penicillin was continued in decreasing doses intramuscularly for four or five days.

The number of cases of suppurative pericarditis treated by the intrapericardial instillation of penicillin has not been sufficiently large to permit any valid conclusions concerning the efficacy of this treatment. Undoubtedly, certain patients are cured by this method alone, as is true in cases of empyema thoracis, particularly those with a nonputrid empyema. As has been reported by a number of observers,[21, 22] however, in the treatment of empyema with penicillin one may sterilize the fluid or pus by vigorous therapy, only to see a recurrence within two weeks. In other cases, the persistence of thick pus and fibrin clots, even though sterile, with consequent organization and thickened pleura, renders the expansion of the collapsed lung impossible or at least subject to long delay. It has therefore been recognized that persistent purulent accumulations in the pleura, even though sterile, should have surgical drainage. I have had the same experience in the treatment of empyemas complicating pulmonary resections, and Lynch and I have found this to be true in the treatment of putrid empyema.[23]

If the same situation obtains with regard to intrapericardial penicillin therapy, the patient should sooner or later present evidence of constrictive pericarditis. Patients who have had such therapy should therefore be carefully observed so that their status in this regard may be evaluated.

Urschel et al.[24] discuss 8 cases of acute pericarditis that they observed during a period of three months. They devised a graph to correlate the heart size, total T-wave deflection, total elevation of the RS-T segment, pericardial friction rub and temperature. Judging from the cases observed, there is no sig-

nificant electrocardiographic difference in the pericarditis caused by rheumatic fever, tuberculosis or pneumococcal or meningococcal infection.

Acute pericarditis simulating myocardial infarction is discussed by Wolff.[25] He states that pericarditis unassociated with other diseases may be characterized by pain resembling that of acute myocardial infarction, and that great care must be used in differential diagnosis, since treatment and prognosis of the two conditions are markedly different. In the several cases of acute pericarditis described by the author, the correct diagnosis was established by the careful study of clinical, laboratory, electrocardiographic, teleroentgenographic and fluoroscopic data.

Constrictive Pericarditis

The use of partial pericardiectomy and epicardiolysis in 24 cases of constrictive pericarditis is discussed by Harrington.[26] The two most frequent conditions with which constrictive pericarditis is likeliest to be confused clinically are cirrhosis of the liver and congestive heart failure from intrinsic cardiac disease. In most cases, however, the subjective symptoms of constrictive pericarditis are sufficiently characteristic so that when they are correlated with the physical and laboratory findings a definite diagnosis can be made.

The laboratory findings are extremely valuable in establishing a diagnosis. The venous pressure is elevated above normal. The circulation time of the blood is increased. The tests of hepatic function indicate a varying degree of hepatic damage, which depends to some extent on the duration of the disease. Roentgenologic studies of the heart in cases of constrictive pericarditis usually show it to be normal or smaller than normal. It is never markedly enlarged or segmentally dilated, as in congestive heart failure from intrinsic cardiac disease and hypertensive heart disease. The presence of calcareous plaques in the pericardium is noted frequently. This always suggests a constricting pericardium, but calcium may occur in the pericardium without other findings of constrictive pericarditis.

In the 24 cases reported by Harrington, the operative mortality was 25 per cent. Of the 18 patients who recovered from operation, 9 were considered cured in the sense that subjective symptoms were relieved and the patient was able to resume his former occupation or to engage in some other useful one. The improvement in 2 more cases had approximated a cure. In 2 other cases in which operation was recent, improvement had been progressive. Thus, 13 of the 24 patients may eventually be considered as cured. Of the remaining 5 patients who recovered from the operation, 2 had shown moderate improvement and 3 had died. Two of these deaths were due to continuation of the disease, and 1 was due to pneumonia.

A vexatious problem that continues to confront the thoracic surgeon is the one presented by the patient who has the symptoms of progressive cardiac compression resulting from active tuberculous pericarditis. The evidence that has accumulated in recent years suggests that in many cases constrictive pericarditis is caused by tuberculosis, but in many cases of tuberculous pericarditis, after the subsidence of the effusion, the onset of progressive compression of the heart becomes evident while the tuberculous lesion is still active, and long before the patient may be considered to have chronic constrictive pericarditis. Under these circumstances, if operation is undertaken, a grossly thickened pericardium may be resected, but of necessity a layer of organizing fibrinous exudate is left adherent to the epicardium, and little if any improvement results. I have recently had such an experience. Partial pericardiectomy was successfully performed, but after a slight transitory improvement the patient died six weeks after operation from a continuation of her disease. Operation was performed with reluctance, and only because the patient was progressively going downhill, although the cardiac-pericardial shadow was becoming smaller.

Blalock and Levy[27] discussed a series of these patients a few years ago and came to the conclusion that one should operate only when it appears that the patient will die if something is not done. Beck[28] is of the same opinion.

Congenital Deficiency of the Pericardium

Rusby and Sellors[29] describe a case of partial deficiency of the pericardium associated with a solitary bronchogenic cyst, both on the left side, in a girl of ten. Eighty such cases have been reported in the literature, but this is only the third case to be recognized during life, and the only one to be carefully studied at operation. Uneventful recovery followed removal of the cyst. The authors believe it likely that partial left-sided deficiency of the pericardium is due to abnormal development, with premature atrophy of the left duct of Cuvier.

Other Indications for Pericardiotomy

After enumerating the standard conditions demanding pericardiotomy, Neuhof[30] suggests its employment for various other conditions, some of which were encountered in the course of thoracic operations undertaken for noncardiac lesions.

In a number of cases of carcinoma of the lung, there was considerable doubt at exploratory thoracotomy regarding the extent of hilar-node involvement, particularly so when the lung was broadly adherent posteriorly and when the tumor in the lung abutted on the posterior mediastinum. Pericardiotomy and exploration within the sac supplied the information. In another case, a carcinoma of the lung at first appeared to be inoperable because of fixation at the mediastinum and invasion of the pericardium. Exploratory pericardiotomy revealed

the extent of the invasion, and liberal sacrifice of the pericardium permitted successful pulmonary resection. I have used this maneuver on several occasions for similar indications and have found it useful.

In the discussion of Neuhof's paper, Haight[31] cited a case in which he resected a large segment of the pericardium so that he might successfully perform pneumonectomy for a bronchogenic carcinoma that was invading the pericardium. Troublesome herniation of the heart resulted in shock if the patient lay on his back or on the operated side postoperatively. It was necessary to keep the patient inclined toward the contralateral side for some days after operation, but eventually he became stabilized, and three years after operation he showed no evidence of cardiac embarrassment.

Neuhof[30] also suggested incision of the pericardium and retraction of the pulmonary artery mesialward in cases of patent ductus arteriosus complicated by subacute endarteritis, so as to facilitate exposure of the deep surface of the ductus. Jones,[32] however, believes that there is a disadvantage in opening the pericardium in these cases, because the trickle of pericardial fluid obscures the field of dissection.

Graham[33] briefly describes the case of a woman who had a recurrence of a carcinoma of the breast in the pericardium, with massive bloody pericardial effusion that required frequent tapping. By the simple procedure of removing some of the pericardium and permitting the recurring effusion to drain into the left pleural cavity, the patient was kept perfectly comfortable for a period of two years, when she died of further recurrences. Graham emphasizes the simplicity of the procedure and the great comfort that it gave to this patient.

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CASE RECORDS OF THE
MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 32161

PRESENTATION OF CASE

A forty-one-year-old truck driver entered the hospital because of a mass in the left upper quadrant of the abdomen.

Five months before admission the patient was referred to the Out Patient Department for study of an albuminuria that had been discovered during an examination for life insurance. In the clinic his urine was repeatedly negative for albumin, although a trace was found on one occasion. A hard, movable mass was found in the left upper quadrant that extended from near the midline into the left flank and from the costal margin downward for 8 cm. It was nontender and moved with respiration. It had not caused symptoms. The liver border was palpated 3 cm. below the rib margin and was separate from the mass. There were external hemorrhoids, but no visible abdominal veins. Examination of the blood was normal except for a slight eosinophilia (8 per cent). X-ray examination revealed a large soft-tissue shadow overlying the left kidney and displacing it somewhat laterally. The spleen looked normal. There was distortion of the superior calyx of the left kidney following an intravenous pyelogram, but owing to the obscuring shadow, this was by no means definite. A barium swallow failed to indicate esophageal varices. The blood nonprotein nitrogen, total protein and albumin-globulin ratio were normal. Bromsulfalein excretion was normal, and the cephalin-flocculation test was negative. The only significant facts in the past history were that he regularly consumed a half pint of whisky every two days. He had never been out of this country. The patient refused immediate operation.

About four months later he began to have occasional slight, nonradiating pains in the region of the mass, lasting from two to three minutes each. He then entered the hospital for operation.

The physical findings were not changed. Examination of the blood showed 4 per cent eosinophils and was not otherwise remarkable. The urine gave a ++ test for albumin, and a concentration test showed a maximum specific gravity of 1.018. A gastrointestinal series demonstrated a large pressure defect in the lesser curvature caused by a mass that displaced the stomach laterally, downward and forward (Fig. 1); the ligament of Treitz was displaced, but there was no deformity of the duodenum. A barium enema was negative. A cholecystogram showed the gall bladder to be normally outlined. There was no blood in the stool.

An operation was performed on the fifteenth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. WALTER GARREY: Apparently this man was machine-processed in an insurance examination in which a urinalysis was done before a complete physical examination. Albumin was found in the urine, and he was then sent to the Out Patient Department, where physical examination showed a hard movable asymptomatic mass in the left upper quadrant. Perhaps this would be a good time to see the x-ray films.

FIGURE 1.

DR. JAMES R. LINGLEY: This is a plain abdominal film showing the mass, which fills almost the entire left upper abdomen. This is a pyelogram showing rather poor filling of the pelvis and calyxes on the left side, probably due to pressure from the mass rather than to intrinsic involvement of the kidney. One can trace the kidney shadow through the mass

quite easily. The colon shows no evidence of intrinsic disease, although the splenic flexure is displaced laterally and somewhat posteriorly by the mass. The gastrointestinal examination shows this large pressure defect on the lesser curvature of the stomach, which involves practically all the lesser curvature and has caused marked displacement of the stomach toward the left. The mucosa over this area looks normal; in other words, I can see no evidence of actual mucosal invasion. The chest plate is negative.

DR. GARREY: I had three questions to put to Dr. Lingley; he has already answered two of them. One was whether the kidney outline was visualized and was entirely separate from the mass. It was. The second question was whether the mucosal pattern was seen clearly down the entire length of the mass. It was. The third was whether there was any calcification in the mass.

DR. LINGLEY: There is none.

DR. GARREY: I am afraid that we do not get much help from the x-ray studies. There was a mass, but I am not even sure what organs were involved. The mass was contiguous to the kidney, liver, spleen, retroperitoneal tissues and root of the mesentery. According to the symptoms, it was increasing in size rather slowly. I cannot imagine any infectious process that would behave this way, with the possible exception of hydatid cyst. This man had not lived in Iceland or the Antipodes, and despite the slight eosinophilia, I do not believe that we can hazard such a diagnosis. The picture certainly does not fit that of a syphilitic liver, so I think we have to assume that the mass was on a congenital or neoplastic basis. Apparently the men in the Out Patient Department thought that the patient possibly had cirrhosis and that the mass might have been derived from the liver, perhaps a primary liver-cell neoplasm. The majority of hepatomas and cholangiomas arise in a cirrhotic liver; I think the figure is around 85 per cent. Is that correct?

DR. TRACY B. MALLORY: Around here.

DR. GARREY: But we have no evidence of cirrhosis. As a further interesting exercise in trying to arrive at a diagnosis, a peritoneoscopy might have been done and even a gastroscopy. A peritoneoscopy probably would not have shown the tumor directly, since it appears to have been located behind the stomach and lesser omentum and mesocolon.

DR. MALLORY: A peritoneoscopy was done. Would you like to have the information?

DR. GARREY: Peritoneoscopy would be worth while on the remote chance that this man had dissemination of a neoplasm and also to rule out the liver disease, but I venture to guess that it was not helpful in telling anything about the primary tumor.

DR. MALLORY: The gall bladder was visualized. The peritoneal cavity appeared normal, except in the left upper quadrant, where a mass could be seen just to the left of the greater curvature, between it and the spleen. The latter could not be visualized. The mass had a grayish-blue tinge, was covered with thin omentum and appeared to be cystic. It was not attached to the abdominal wall. There was no lobulation.

DR. GARREY: Very wisely no biopsy was done, since if it were a cystic mass they did not want to spill material freely into the peritoneal cavity. I am very much interested in the physical finding of mobility, which makes the diagnosis all the harder to me. They speak of the mass as being movable in the first examination, and also that it moved with respiration, and again in the examination in the hospital four months later they mention that the mass was movable. The most probable mass in this area that has this characteristic position, size and symmetry is a cyst of the pancreas. We get no lead from the history. There was no trauma, and no biliary tract disease, which might have predisposed to a pseudocyst. A rather small percentage of pancreatic cysts are movable. The nearer they are to the tail of the pancreas and the more ptotic the patient, the likelier the mobility. Pancreatic cyst seems to be a strong possibility, although the patients with pancreatic cysts whom I have seen have had more pain than this man appeared to have, provided that the tumors had reached this size. I assume that the albuminuria was due to pressure on the left renal vein and kidney. This has been reported in an occasional case.

I do not believe that we are dealing with a particularly malignant situation because the man had no constitutional symptoms or signs. He did not appear to have run downhill after this five-month period. If he had a solid neoplasm of a disseminating, highly malignant character, I should expect that some other finding would have been manifest by this time. I do not believe that, with the mobility that they have emphasized, a retroperitoneal neoplasm, a sarcoma, is likely, or a large fibrosarcoma. This does not appear to have had the character of a lymphomatous tumor: it was not nodular, and it appeared symmetrical and cystic, both in the x-ray films and at the time of peritoneoscopy. There are rare tumors that may arise in the space behind the stomach — hemangioendothelioma, dermoid cyst, a cyst containing sebaceous material and tumors of neurogenic origin. I am driven away from virulent neoplasm and incline more and more to the supposition that this was a relatively benign lesion occurring in tissue at the root of the mesentery or possibly in the pancreas. Fifteen days were spent in studying this man in an attempt to make a diagnosis. They also were groping for any concurrent disease that might have given a lead to the diagnosis, but they wisely ruled it out.

My first hunch is pancreatic cyst, either a pseudocyst or a cystadenoma. My second hunch is some

rare but relatively benign tumor occurring in the same area, possibly a cyst of congenital origin high in the mesentery, although the location was higher than it usually is in cases of this sort. I think that they probably had to end up by doing what is recommended by one of the late Dr. G. W. W.Brewster's dictums, which some of you may remember. He urged, that if one found trouble, to cut over it.

DR. MALLORY: Dr. Volwiler, you studied this patient on the ward. Have you any comment?

DR. WADE VOLWILER: I saw him with the third-year medical students in the Out Patient Department, and we reached no definite decision. We thought that the likeliest diagnosis was pancreatic cyst.

DR. CLAUDE E. WELCH: It took Dr. Garrey only twenty minutes to make the wrong diagnosis, but it took us over two weeks. The albuminuria, which Dr. Garrey was somewhat interested in, turned out to have no significance. So far as the tumor was concerned, we thought that it was a pancreatic cyst, because it was somewhat spherical and therefore probably represented encysted fluid rather than a solid tumor. On entering the abdomen we found that this was a solid tumor tightly attached to the wall of the stomach. As a matter of fact it was impossible to separate the stomach from the tumor. There was no attachment elsewhere. We therefore had to proceed with a subtotal gastric resection. We sent for a pathologist to look over the specimen as soon as it was removed, because we were interested in the fact that large, probably hyperplastic, lymph nodes extended up the left gastric artery. We wanted to be sure that the line of resection, which was close to the esophagus, was free of disease. The pathologist's opinion was that it was a benign tumor.

CLINICAL DIAGNOSIS

Cyst of pancreas.

DR. GARREY'S DIAGNOSIS

Cyst of pancreas.

ANATOMICAL DIAGNOSIS

Neurinoma of stomach.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The tumor was a benign spindle-cell tumor of the stomach. There might be some argument whether to classify it as a leiomyoma or as a perineurial fibroma. I personally believe that it was the latter. Its apparently cystic character was due to extreme vascularity. It was so vascular that when we first saw the section the possibility of angioma suggested itself to our minds. There was no extension of the tumor beyond the point of actual attachment to the gastric wall, and I think that there is no question that cure has been achieved.

DR. WALTER BAUER: In retrospect, would the radiologist interpret the films any differently?

DR. LINGLEY: I should still think that it was a pancreatic cyst.

CASE 32162

PRESENTATION OF CASE

A fifty-year-old man entered the hospital because of stomach pain.

Fourteen months before admission the patient first began to have crampy midepigastric pain that came on three or four hours after each meal. He consulted his physician, who obtained a gastrointestinal series that was said to have demonstrated a stomach ulcer. He was put on a liberal, low-fat, bland diet and given "powders and white liquid" to take between meals. On this regime he obtained almost complete relief for seven or eight months, when he had a recurrence of postprandial pain, which persisted despite the powders but was relieved by food. One month before admission he had another gastrointestinal series, following which he was told that he would have to have his stomach removed. He belched frequently and felt epigastric fullness and vague uneasiness after meals. On a few occasions he induced vomiting because of distressing fullness. The bowel movements were regular. No bloody or tarry stools were noticed.

The physical examination was negative except for a walnut-sized indirect inguinal hernia.

The temperature, pulse and respirations were normal; the blood pressure was 110 systolic, 70 diastolic.

The hemoglobin was 15.8 gm. per 100 cc. The urine was normal. A gastric analysis showed no free hydrochloric acid in the first specimen, and 27 and 15 units, respectively, in the second and third specimens.

X-ray examination of the stomach showed a marked degree of spasm of the antrum. At the midportion of the lesser curvature there was a shallow collection of barium, which might have represented a flat crater. The first portion of the duodenum was constantly deformed at its apex; no crater was found in that region. There was a 1-cm. diverticulum projecting medially from the second portion of the duodenum. The heart and lungs were normal by x-ray examination.

An operation was performed on the fifth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. WADE VOLWILER: I infer that this man was moderately obese, since the only reason for giving a low-fat diet to a patient with simple ulcer is to avoid a gain of weight during a program of frequent feedings.

The center of interest here lies in the identity of this vague questionable lesion in the lesser curvature, and I think that we should have an x-ray opinion first. I should like to ask several specific questions: Can we assume that this duodenal deformity represents the crater of a benign ulcer at some time in the past? Can this vague lesion in the lesser curvature be interpreted in one of three ways—a scar following the healing of an ulcer in that area, a flat shallow crater or just a collection of barium between hypertrophied gastric rugae?

Dr. James R. Lingley: I did the examination on this particular patient, and those were the exact questions that I had in mind while doing it. I had great difficulty in determining whether or not this lesion in the lesser curvature was a crater. The most remarkable thing about the stomach was the extreme spasm of the antrum. Trying to find some cause for the spasm, I studied the area on the lesser curvature extremely carefully and found a lesion 1.5 cm. in size where the barium tended to puddle out and form a shallow crater.

Dr. Volwiler: Was there rigidity over that area?

Dr. Lingley: That was difficult to determine because the stomach was high, lying under the costal border, and we could not palpate it. There was no peristalsis in that region. I thought that it was a crater, probably an extremely shallow one, representing an almost healed ulcer. The patient showed a constant deformity of the apex of the duodenum and a small diverticulum in the second portion.

Dr. Volwiler: The duodenal picture probably represents the healed stage of a benign ulcer.

Let us go back to the present illness, which began with the typical symptoms of peptic ulcer, and which disappeared almost completely in eight months on a fairly satisfactory ulcer regime. I am going to assume that during that time the original ulcer had healed almost completely, if not completely. We do not know where the ulcer was, but it could have been either a duodenal or a gastric ulcer. I think that we should take the patient's statement at face value and assume that it was a gastric ulcer and that it had probably originated at the site of the present defect. During the two months before entry there was a sudden change of symptoms, which is significant. Diffuse hypertrophic gastritis could have produced the recent symptoms of this man and might have been difficult to demonstrate by x-ray. It can occur in a localized form, but it is seldom observed on the midportion of the lesser curvature and the localized form usually does not give rise to such extensive symptoms. I believe that the development of such a process, leading to the symptomatology that existed here during the time that the patient was following a fairly good regime, is highly unlikely, and I therefore shall discard it. I shall also discard a scar as an explanation of the lesion, because that, of course, could not explain the symptoms.

We should perhaps mention cholecystitis, cholelithiasis and even subacute recurring mild pancreatitis as an explanation of the symptoms. There is no evidence for them, however, and we should focus our attention on the stomach. I am not sure whether there was an ulcer crater. But regardless of that fact, I think that, in view of the symptoms, the most logical explanation for the lesion is a small, slowly growing, infiltrating carcinoma. I say that in spite of the facts that this patient probably was moderately obese, that he had previously had a benign lesion in the duodenum, that the stomach produced free acid and that the x-ray defect was questionable. This patient certainly should have been operated on. A preoperative gastroscopy might have given valuable information, but regardless of what was found, I believe that the stomach should have come out. This may have been one of those occasional extremely difficult cases in which neither the surgeon nor the pathologist on gross examination can decide what the lesion is. It may have been only the microscopical examination that revealed the true identity of the lesion.

Clinical Diagnosis

Gastric ulcer.

Dr. Volwiler's Diagnoses

Carcinoma of stomach.
Old benign duodenal ulcer.

Anatomical Diagnosis

Carcinoma of stomach.

Pathological Discussion

Dr. Tracy B. Mallory: The patient was operated on, and a subtotal gastric resection was performed. We found on the lesser curvature a localized area, slightly over 2 cm. in diameter, where the mucosa appeared thicker and firmer than elsewhere, with a somewhat different translucence and color; at one margin there was a shallow erosion. The wall of the stomach was thickened and indurated under this area of altered mucosa. The gross appearance, however, was essentially that of a healed ulcer rather than of a neoplasm. When the microscopic preparations came through it became evident that the entire area of altered mucosa showed a carcinoma in situ. At one single spot tumor cells had invaded the submucosa, and two or three glands had penetrated a short distance into the muscularis. Several small lymph nodes were examined. All were negative, as one would expect with such an early lesion.

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THE INADEQUACIES OF MEDICAL CARE

II. Those Concerning Its Distribution and Cost

Of utmost interest at the moment in matters pertaining to medical care are its distribution and cost. It is a well recognized fact that many persons fail to receive adequate medical care either because it is unavailable or because they are of the opinion that they cannot afford it. At times this is inexcusable, since the means are at hand provided that the person knows how to obtain them. On the other hand, in the majority of cases there is a just complaint. In many areas facilities are lacking, in others they are inadequate, and in all certain fees for medical care are out of all proportion with the income of the patient.

Obviously legislation should be directed toward the relief of sections that lack facilities for medical care. Hospitals and health centers should be constructed and equipped by the state, with the assistance of federal grants-in-aid, and these in turn would have a tendency to attract properly qualified physicians. To be sure, the economic status of any particular section may be such that the physician is unable to obtain a living wage, but that again is a responsibility of the state, which should guarantee a reasonable income by supplementing what he is able to earn with a salary or retaining fee.

Inadequate care involves poorly trained physicians, those who had proper training but have failed to keep up with medical advances, a scarcity of qualified specialists and lack of proper hospital facilities. The first two concern the quality of medical care and the last has to do with hospital rather than medical care, all of which will be discussed in subsequent editorials. Lack of well trained specialists in any given section is largely a matter of insufficient incentive for the specialist to practice there; physicians of this type naturally tend to congregate in large or moderate-sized cities, where well equipped hospitals are located and where they are satisfactorily paid for their services either by salaries or by fees. To persuade them to settle in sparsely settled areas or in those of low economic status again will require state aid.

The matter of cost of medical care naturally receives the topmost attention from the public. By and large, the indigent, particularly those who reside in or near urban areas, now receive reasonably good medical care with little or no cost, and those with low to moderate incomes are capable of handling the expense of minor illness with slight financial inconvenience. But the consequences of a major medical or surgical illness, including loss of wages, is a catastrophe for which few of the latter group have provided.

No doubt much needs to be done to improve the medical care of the indigent in areas other than urban. Many of the handicaps to this type of care would be removed if hospitals and health centers were made available, the cost being met by state funds, with the assistance of federal grants-in-aid.

A great deal has been written concerning the high cost of medical care. There is no doubt that this expense has risen tremendously in recent years, but

so, too, has that for almost everything else. On the other hand, what appear to be large fees do not necessarily mean a high net income to the physician. His training, particularly if he is a qualified specialist, represents a considerable capital outlay, which often must be repaid over a period of years; furthermore, his actual cost of successfully practicing medicine is many times larger than that of the old family doctor. Hospitalization and expert nursing care also add appreciably to the expense of a serious illness, but no one can deny that the patient who is obliged to undergo a gastrectomy is better off in a modern hospital than he would be if he were operated on lying on a door in the kitchen or back parlor. No doubt the cost of serious illness has risen, nor does there seem to be any logical way in which it can be appreciably lowered in any individual case. All of which means that persons in the low-income group and even in the moderate-income group must set aside money in anticipation of catastrophic illness: in other words they must insure against it just as they insure against fire or burglary. Compensation for loss of wages due to illness might well be included among the benefits provided by the Social Security program.

It seems likely that the cost of good medical care can be lowered by group practice. In the obscure case, the opinions of various specialists can obviously be obtained for less actual cost and with less inconvenience to the patient if these men have banded together in a group than if they are practicing as individuals. Outstanding examples of this type of organization are the nationally known large private clinics and those connected with teaching hospitals, and there are hundreds of small groups scattered throughout the United States, particularly in the Mid-West and South. This type of practice has much to offer, and it will probably become more popular in the near future than it has been in the past.

In summary, the inadequacies of the distribution of medical care are largely the concern of government, although ignorance of the public concerning available facilities is far from a negligible factor. The high cost of medical care can undoubtedly be somewhat lowered by group practice, but there appears to be no way of eliminating the expense of catastrophic illness, other than by insuring against it.

MASSACHUSETTS MEDICAL SOCIETY
POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

TEACHING HOSPITALS

In a recent address, Dr. Alan Gregg,[1] director for the medical sciences, Rockefeller Foundation, discussed the place of the teaching hospital in the training of medical students and nurses, for the specialized and advanced training of physicians and for the advancement of medical knowledge. In addition, he emphasized what may be of equal or even greater importance to the public who supports these hospitals, namely, that the teaching hospital provides the best type of care for the patient. In speaking about the patients whose cases are studied by medical students and their teachers, Dr. Gregg said in part:

> What an amazing misconception about this procedure is rampant among most laymen! They think that when they consent to be part of a demonstration in a clinic, for teaching purposes, they are conceding something of their comfort and well-being to the professional zeal of young doctors. The exact opposite is true. When a patient consents to this procedure, his case receives the most careful, the most accurate and most modern treatment. Actually, it has all the advantages of an early consultation. So few people understand this. So many . . . especially of the middle class income group . . . ought to realize this. They should know that to consent to be used for teaching is to display the most enlightened self-interest.

In a comment on President Truman's special message to Congress advocating compulsory national medical insurance, the Committee on Medicine and

the Changing Order of the New York Academy of Medicine[2] made some pertinent suggestions. To raise the quality and to round out the scope of medical care extended under any system of payment, the committee favors not only a number of revisions in medical education but also the gradual extension of group practice. In this connection, it is stated:

> It is essential, moreover, that medical group practice units be organized also in relation to teaching hospitals, since an extension of prepaid medical care to all people of low income must inevitably have important repercussions on medical education and research. The medical schools, for their part, must change their methods if they are to train doctors for a new form of medical care; and no form of medical care can afford to neglect the advantages to the patient of teaching and research.

That the teaching hospital must provide the best type of medical care for the patients can readily be appreciated on the briefest reflection. The patient whose case is used for teaching *must* receive the best of modern care. Everybody from student through intern and resident to visiting physician must be on the alert and aware of the up-to-date methods of diagnosis and treatment. The alternative is to lay oneself open to censure or to lose dignity and respect.

Perhaps the greatest possibilities for abuse by the medical profession exist in the practice of surgery or of the surgical specialties. In small hospitals and in strictly private hospitals, or even in the large public hospitals, if there is not a feeling of responsibility on the part of the surgeon to justify every step, and also the willingness on his part to take the time and trouble to keep in touch with medical progress, the patients suffer. This, however, is not limited to surgeons. One can readily imagine the abuses that are possible in the care of neuropsychiatric cases by unscrupulous physicians when the management of their cases is not subject to review and justification.

The Veterans Administration, in its recent reorganization, has accepted the concept of the teaching hospital as the only available means of establishing and maintaining the highest quality of medical service. Teachers from medical centers are to be chosen to staff its hospitals on a part-time basis. They are expected to provide the incentive for the resident staff to study their cases adequately, so as to be able to provide the best of medical care. The close association of interns and residents with these teachers will also provide inspiration to the younger men to sustain interest, to follow their cases closely and to correlate their findings and thus be in a position to improve and advance the quality of service that they offer.

In hospitals whose staffs are not closely allied with medical schools, promotions within the staff are based entirely on seniority. It is obvious that when such promotions are made without considering the professional qualities and attainments of the physician the quality of the care that the patients receive is apt to suffer. Naturally, those who would otherwise rise by seniority rather than by attainment are likely to resist any attempt by medical schools to deprive them of their rights to promotion. The sole duty of medical schools is to provide good clinical instruction, and this predicates the best medical care for the patient and the choice of the persons best qualified to give such care. Thus, resistance to change was undoubtedly met by the new heads of the Veterans Administration when many of the less qualified among the senior officers had to be replaced.

Administrators and trustees of public hospitals and their medical staffs would do well to take the cue from the Veterans Administration. Needless to say, the least qualified among the staff members will be relegated to less responsible positions when a medical school affiliating with a hospital is given a free hand in the selection of teachers and a determining role in promotions within the staff. When a hospital staff resists such a move as an encroachment on its prerogatives, it is paramount to an admission either of inferiority or of a lack of interest on the part of its members. In other words, such an attitude must be interpreted as indicating either that they are unsuited for the positions or that they are not sufficiently interested in the welfare of their patients and of the hospital and in the improvement of medical practice to take the time and to put in the extra effort that is required of the members of the staff of a teaching hospital.

Both the hospital and the medical school have added responsibilities when they become affiliated, and these all accrue to the benefit of the patient.

The hospital must provide the facilities for the best of medical practice, and these are always under the scrutiny of the students and their preceptors. The latter must always be on their toes and keep up to date. Medical schools, on the other hand, have the responsibility of seeing that they make the best choice and that they recommend promotions solely on the basis of merit and achievement.

REFERENCES

1. Gregg, A. Trends in medical education. A chapter in *The Doctors Talk It Over*. Vol. II. New York: Lederle Laboratories, Incorporated, 1945.
2. Statement. Medicine and the changing order. *J. A. M. A.* **130**: 94, 1946.

MASSACHUSETTS MEDICAL SOCIETY

ANNUAL MEETING OF THE COUNCIL

The annual meeting of the Council will be held in the Georgian Room of the Hotel Statler, Boston, on Tuesday, May 21, 1946, at 7:00 p.m. This meeting will be preceded by the Cotting supper which will be served in Parlors A and B of the same hotel at 6:00 p.m.

Business:

1. Presentation of record of the stated meeting of the Council held February 6, 1946. To be published in the *New England Journal of Medicine*, issue of May 2, 1946.
2. Reports of standing and special committees.
3. Election of officers and orator.
4. Appointment of committees for ensuing year.
5. Such other business as may lawfully come before the meeting.

MICHAEL A. TIGHE, *Secretary*

SUBCOMMITTEE ON VETERANS' AFFAIRS, POSTWAR PLANNING COMMITTEE

In the course of its investigation of the needs of medical officers who had returned from the armed forces, the Subcommittee on Veterans' Affairs soon stumbled on the serious problem of early telephone listing for doctors reopening their offices. The committee pursued this subject as soon as possible in order to get early action thereon. A preliminary report of the committee's findings is as follows:

The next telephone directory will be published for the Boston District in December. Owing to the paper shortage this is the earliest date that the Government will permit publication of a new directory. The New England Telephone and Telegraph Company is fully aware of the seriousness of this problem, not only for doctors but also for other professional men. It has canvassed means to solve it and has taken the following steps:

(1) A leaflet will accompany the April telephone bills urging subscribers to make use of Information Service for numbers that they cannot find and pointing out that since the last directory was published there has been a gain of 72,000 phones in the Boston area (225,000 in the total area served).

(2) It has expanded Information Service and is continuing this expansion as fast as new operators are trained. In the Boston area there will eventually be an increase of 15 per cent (128 operators) in the personnel of this service.

(3) Beginning about April 1, the company will stress the use of Information Service in advertising especially in the daily papers. This will be a regular campaign, and the company accepted the suggestion to stress the use of this service in the advertising in connection with the phrase "your doctor" or "your attorney who has returned from the services."

(4) The company welcomes any suggestion as to how to make doctors' numbers more readily available to the public, subject to the limitations outlined above. At the present time every number is in the hands of Information Service within twenty-four hours of the installation of the telephone.

(5) Doctors can help this campaign by telling their patients to use Information Service for numbers they fail to find in the directory.

The company has been most courteous and patient and has explained to us in detail many of the difficulties involved and the reasons that led them to adopt this as the only solution to the present dilemma.

G. PHILIP GRABFIELD, *Chairman*

TREASURER'S REPORT COVERING REFUND DISTRIBUTION

The Treasurer of the Massachusetts Medical Society makes the following report regarding the refund to district societies for 1946:

The Council voted to distribute the sum of $4000 to district societies. The total number of payments of annual dues received by the Treasurer by March 1, to be counted for the refund, was 3965. Therefore the refund to the district societies for each paid fellow is $1.009.

The following table gives the number of payments, as of March 1, and the refund to each district as of April 1:

DISTRICT	NUMBER REPORTED PAID	REFUND
Barnstable	39	$39.32
Berkshire	104	104.88
Bristol North	57	57.48
Bristol South	155	156.36
Essex North	150	151.31
Essex South	217	218.91
Franklin	35	35.29
Hampden	260	262.30
Hampshire	61	61.51
Middlesex East	114	114.99
Middlesex North	102	102.88
Middlesex South	810	817.25
Norfolk	699	705.26
Norfolk South	107	107.92
Plymouth	114	114.99
Suffolk	532	536.75
Worcester	332	334.95
Worcester North	77	77.65
	3965	$4000.00

In 1945, for comparison, the total number of payments for the refund was 3297.

ELIOT HUBBARD, JR., M.D., *Treasurer*

MEDICOLEGAL ABSTRACT

Relation of Patient and Physician: Liability for malpractice. The plaintiff alleged that the defendant, a dentist, had broken off a drill and hypodermic needle in his jawbone, that these were the cause of his illness and considerable medical expenses to which he had been put, that the defendant knew that the drill and the needle were there and

that he had fraudently concealed the facts from the plaintiff.

The defendant made a motion to dismiss by reason of the acts having taken place more than two years before the suit was begun.

The Arizona court held that the dentist had a duty to disclose the facts to the plaintiff and that his failure to do so was such fraud as to prevent the running of the Statute of Limitations until the facts fraudulently concealed became known. — (*Acton v. Morrison*, 155 Pac. [2d] 782, Jan. 29, 1945, Arizona.)

This case may be distinguished from a Massachusetts case in which it was alleged that the defendant, a physician, left gauze in the plaintiff during an operation performed more than two years before the action was brought. The gauze was not discovered until shortly before the suit was begun. There was no allegation that the doctor knew the gauze had been left in. The Supreme Judicial Court in holding that the action was barred by the Statute of Limitations said that there was no showing that the defendant had fraudulently concealed the facts.—(*Capucci* v. *Barone*, 266 Mass. 578 [1929].)

It is noteworthy that the court in the first case considered the relation of dentist and patient such a fiduciary relation that a mere failure to disclose without any affirmative act of concealment was sufficient to constitute fraud.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CURED CANCER CLINICS

At the moment, fourteen Cured Cancer Clinics are scheduled to be conducted during the month of April. These clinics represent a special form of teaching clinic in which only cured cases will be shown. Such teaching clinics, held at the places and meeting of the regular state-aided cancer clinics, are designed for the purpose of advising the physicians of the community regarding cancer, its symptoms, diagnosis and treatment.

The clinics will be held at Beverly, Boston (Boston Dispensary and Beth Israel Hospital), Brockton, Fall River, Fitchburg, Gardner, Greenfield, Lawrence, New Bedford, Pittsfield, Westfield (State Sanatorium), Worcester and Wrentham (Pondville Hospital).

Information regarding the dates and times of the Cured Cancer Clinics will be provided at the regular state-aided cancer clinics or at the local district health offices.

MISCELLANY

NEW PSYCHIATRY PROGRAM AT BOSTON UNIVERSITY

With the appointment of Dr. William Malamud as professor of psychiatry and chairman of the Department of Psychiatry at Boston University School of Medicine, the University will undertake a program of undergraduate instruction in this field that will meet the need for providing medical students with a more extensive training.

In making the announcement of the appointment of Dr. Malamud, who is now director of research at the Worcester State Hospital, President Daniel L. Marsh said that the School of Medicine has recognized for a long time that the psychiatry training offered today to students in medical schools is generally inadequate. An anonymous gift of $30,000 from a long-time friend of the University has now made possible the expansion of this part of the school's instructional program.

Under the direction of Dr. Malamud, the University will inaugurate a program of undergraduate training in psychiatry that will be carried on throughout all four years of the medical-school course. Special emphasis will be placed on giving the students an increased opportunity to receive training in the diagnosis and management of the neuroses, which are now recognized as constituting a large part of all medical practice.

CARE OF NONSERVICE-CONNECTED ILLNESS

Treatment in private hospitals, at government expense, of male veterans whose illnesses are not service connected cannot be authorized by the Veterans Administration, according to officials of the Boston Branch Office of the Veterans Administration. The statement was made to correct misinformation among both veterans and private-hospital authorities. Veterans whose disabilities are service connected may receive private-hospital treatment at government expense. Except in emergency cases, however, the veteran must make arrangements for such private-hospital care with the Veterans Administration prior to entering the institution.

In discussing nonservice-connected cases, the officials point out that arrangements may be made for treatment of women veterans in private hospitals at government expense if Veterans Administration hospital facilities are not available. Male veterans with nonservice-connected illnesses may be treated at Veterans Administration hospitals at government expense if beds are available in addition to those required by veterans whose disabilities are service connected.

BOOK REVIEWS

The Diagnosis and Treatment of Acute Medical Disorders. By Francis D. Murphy, M.D. 8°, cloth, 503 pp., with illustrations. Philadelphia: F. A. Davis Company, 1944. $6.00.

There are acute episodes and emergencies in medicine as well as in surgery. The disturbing problem that confronts the physician in such acute cases is one of correct diagnosis; the rest of the battle is to follow a certain routine treatment. In this book Dr. Murphy has grouped a number of such emergencies under various headings and has dealt with them masterfully. It is really a volume on differential diagnosis, although the treatment in each case is not neglected. But it is the former that presents the major problem — things are not always what they appear to be on the surface.

The discussions in general are well presented and adequate. In a few places they appear to lack somewhat in detail, but some curtailment was undoubtedly necessitated for the sake of brevity. A great deal of material and up-to-date information has, however, been crowded into this volume. Some of the discussions on differential diagnosis are excellent for clarity and presentation and reveal the true teacher. Unfortunately, for the book-buyer, medical progress is so rapid that often by the time a volume is published new treatments and remedies have come to light; for example, in the treatment prescribed for subacute bacterial endocarditis, the author does not include massive doses of penicillin, for which recent reports claim great promise. Even in this day of refresher courses, here is a valuable volume that is stimulating and refreshing.

Medical Care and Health Services for Rural People: A study prepared as a result of a conference held at Chicago, Illinois, April 11-13, 1944. 8°, paper, 226 pp., with 12 charts. Chicago: Farm Foundation, 1944. Single copies, each $1.00; five or more copies, each 60 cents.

This book is the report of a conference held at Chicago on April 11–13, 1944, which was sponsored by the Farm Foundation for the purpose of discussing medical care and health services for rural people. Representatives of many farm organizations, governmental agencies, medical organizations, universities and voluntary organizations comprised

most of the discussant group. Every phase of the problem was given consideration. Views expressed were as divergent as the opinions of the participants.

A committee comprised of representatives of the Farm Bureau, the Farmers Union, the Grange, and the Southern Tenants in Farmers Union submitted the following recommendations:

1. That the governor of each state be asked to appoint a committee or commission to study the problems of rural health and medical care in the respective states.
2. That farm organizations be encouraged to continue study and experimentation in the field of health service, drawing upon local and national professional and civic groups, such as the associations of hospitals, physicians, public-health organizations, medical colleges and other groups.
3. That the foundation stimulate the establishment of scholarships for farm boys and girls who will study medicine and nursing and return to rural areas for practice.
4. That the foundation establish a national committee on rural health by providing technical services and information and by inviting the national farm organizations to name representatives to serve on the committee.

This book, with its large amount of information, should be of considerable interest to physicians studying medical economic problems.

Medical Uses of Soap: A symposium. Edited by Morris Fishbein, M.D. 8°, cloth, 182 pp., with 41 illustrations. Philadelphia: J. B. Lippincott Company, 1945. $3.00.

Erasmus Wilson in the middle of the nineteenth century popularized the bath by his writings for laymen on the care of the skin. Since then soap has become increasingly popular, and as one of the collaborators of this book states, "There is a trite maximum to the end that the per-capita consumption of soap is a yardstick of a nation's degree of civilization." When the busy dermatologist reviews his practice, however, and notes the large number of soap-sensitized individuals he has to treat, he wonders whether or not the per-capita consumption of soap in America is not too excessive, as a result of the propaganda of soap manufacturers.

This treatise on soap is timely but must be read rather carefully and weighed in the balance. As stated in the preface, a book of this type — written by a group of collaborators — is bound to have a certain amount of duplication. This is noted not only in the discussion but also in the illustrations. Instead of repeating illustrations, pictures of other dermatoses would have been more appropriate. The first few chapters on the chemistry of soap are excellent; in fact, the book is so well done that it is difficult to find fault.

A few over-statements might be mentioned, such as on page 63, where it states acne due to chlorinated compounds can be prevented and aided by soap and water. This does not agree with the reviewer's experience in treating such cases. With reference to page 69, most plating procedures, such as nickel plating, are done in acid baths. After reading the discussion on synthetic detergents on page 84, one would think that sulfonated oils were a distinct entity, whereas they are probably the commonest of synthetic detergents. These remarks are quoted merely to show what an excellent symposium this collection of articles forms. This is a book that should be read by everyone interested in soap, and should be a valuable addition to the library of the physician, the student and the layman.

NOTICES

ANNOUNCEMENTS

Dr. Robert J. Cataldo announces the opening of his offices for the practice of internal medicine at 820 Main Street, Waltham, and 475 Commonwealth Avenue, Boston.

Dr. Trygve Gundersen wishes to announce his return to the practice of ophthalmology at 101 Bay State Road, Boston.

Dr. Sumner D. Liebman announces his return from military service and the opening of an office for the practice of ophthalmology at 115 Bay State Road, Boston.

MASSACHUSETTS PHYSIOTHERAPY ASSOCIATION

The Massachusetts Physiotherapy Association, Inc. porated, which is the Massachusetts chapter of the America Physiotherapy Association, will gladly furnish a directo of its members to any member of the medical professio Requests should be addressed to Mrs. Dorothy V. Warre Physical Therapy Department, Massachusetts Gene Hospital, Fruit Street, Boston.

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

The regular spring meeting of the New England O Laryngological Society will be held at the Massachuse Eye and Ear Infirmary on Wednesday, May 8.

AMERICAN COLLEGE OF CHEST PHYSICIANS

The next oral and written examinations for fellowship the American College of Chest Physicians will be held at S Francisco on June 29. Applicants for fellowship in the C lege who plan on taking the examination should communic with the Executive Secretary, American College of Ch Physicians, 500 North Dearborn Street, Chicago 10.

The twelfth annual meeting of the College is schedu to be held at the Sir Francis Drake Hotel, San Francis on June 29 and 30 and July 1 and 2.

BOSTON GASTROENTEROLOGICAL SOCIETY

A meeting of the Boston Gastroenterological Society be held at the Harvard Club of Boston on Wednesd April 24, at 12 m. Dr. Reginald Fitz will deliver an addr on the subject "Appendicitis and Its Complications." Lun eon will be served free to members and a nominal cha will be made for guests. Reservations may be made telephoning the secretary, Dr. C. W. McClure at KENm 3200.

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

The annual meeting of the Massachusetts Society of Ex ining Physicians will be held on Wednesday, May 8, in Colonial Room of the Copley Plaza Hotel, Boston.

Dinner is scheduled for 6:30 p.m., to be followed b round-table discussion of the Wagner–Murray–Dingell B with Dr. Joseph S. Lawrence, chief public relations office the American Medical Association at Washington, D. and Dr. Allan M. Butler, of the Massachusetts Gen Hospital, as the chief speakers.

Because of the interest in the subject to be discussed, society will welcome nonmembers who may wish to atte Reservations for the dinner may be made by writing to M Anne Rodman, Assistant Secretary, 157 Fourth Str Medford, Massachusetts.

NEW YORK ACADEMY OF MEDICINE

The Nineteenth Graduate Fortnight of the New Y Academy of Medicine will be held October 7 through Oct 18, the subject being "Tumors." The program incl evening lectures, morning panel discussion, scientific exh and demonstrations at the Academy and afternoon cli at leading hospitals of New York City. Complete progr will be mailed to physicians on request.

A physician who is not a fellow of the Academy may se registration by sending his name and address, accompa by a check for five dollars, to the Secretary of the Grad Fortnight Committee, 2 East 103rd Street, New York New York. Medical officers of the United States Ar Navy and Public Health Service, on active duty, will be mitted to all sessions without a registration fee.

(*Notices continued on page xvii*)